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NEWS	4	DEC 08	INPADOC: Legal Status data reloaded
NEWS	5	SEP 29	DISSABS now available on STN
NEWS	6	OCT 10	PCTFULL: Two new display fields added
NEWS	7	OCT 21	BIOSIS file reloaded and enhanced
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NEWS	10	DEC 08	CABA reloaded with left truncation
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NEWS	16	DEC 19	CROPU no longer updated; subscriber discount no longer available
NEWS	17	DEC 22	Additional INPI reactions and pre-1907 documents added to CAS databases
NEWS	18	DEC 22	IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
NEWS	19	DEC 22	ABI-INFORM now available on STN
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NEWS	21	JAN 27	A new search aid, the Company Name Thesaurus, available in CA/CAPLUS
NEWS	22	FEB 05	German (DE) application and patent publication number format changes
NEWS EXPRESS			DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003
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=> file caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
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FILE COVERS 1907 - 6 Feb 2004 VOL 140 ISS 7  
FILE LAST UPDATED: 5 Feb 2004 (20040205/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s TNFR(w)DD

1077 TNFR  
95 TNFRS  
1102 TNFR  
(TNFR OR TNFRS)  
7892 DD  
1630 DDS  
9486 DD  
(DD OR DDS)

L1 2 TNFR(W)DD

=> d 1-2

L1 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN

AN 2001:190739 CAPLUS  
DN 134:339415  
TI Structure-activity relationship of the p55 TNF receptor death domain and its lymphoproliferation mutants  
AU De Wilde, Gert; Murray-Rust, Judith; Boone, Elke; Olerenshaw, Dionne; McDonald, Neil Q.; Ibanez, Carlos; Haegeman, Guy; Wollmer, Axel; Federwisch, Matthias  
CS Department of Molecular Biology, University of Gent-VIB, Belg.  
SO European Journal of Biochemistry (2001), 268(5), 1382-1391  
CODEN: EJBCAI; ISSN: 0014-2956  
PB Blackwell Science Ltd.  
DT Journal  
LA English  
RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 2000:492797 CAPLUS  
DN 133:251048  
TI Mutational analysis and NMR studies of the death domain of the tumor necrosis factor receptor-1  
AU Telliez, Jean-Baptiste; Xu, Guang-Yi; Woronicz, John D.; Hsu, Sang; Wu, Jing-Lun; Lin, Laura; Sukits, Steven F.; Powers, Robert; Lin, Lih-Ling  
CS Department of Musculoskeletal Science and, Wyeth Res., Cambridge, MA, USA  
SO Journal of Molecular Biology (2000), 300(5), 1323-1333  
CODEN: JMOBAK; ISSN: 0022-2836  
PB Academic Press  
DT Journal  
LA English  
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s (tumor necrosis factor death domain)  
297291 TUMOR  
122350 TUMORS  
337436 TUMOR  
(TUMOR OR TUMORS)  
88862 NECROSIS  
2 NECROSISES  
88864 NECROSIS  
(NECROSIS OR NECROSISES)  
814930 FACTOR  
717946 FACTORS  
1288576 FACTOR  
(FACTOR OR FACTORS)  
105566 DEATH  
8785 DEATHS  
111910 DEATH  
(DEATH OR DEATHS)  
217943 DOMAIN  
116303 DOMAINS  
275587 DOMAIN  
(DOMAIN OR DOMAINS)  
L2 6 (TUMOR NECROSIS FACTOR DEATH DOMAIN)  
(TUMOR (W) NECROSIS (W) FACTOR (W) DEATH (W) DOMAIN)

=> d bib, abs 1-6

L2 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 2003:886061 CAPLUS  
DN 139:349546  
TI The death domain kinase RIP1 is essential for tumor necrosis factor alpha signaling to p38 mitogen-activated protein kinase

AU Lee, Thomas H.; Huang, Qiaojia; Oikemus, Sarah; Shank, Jennifer; Ventura, Juan-jose; Cusson, Nicole; Vaillancourt, Richard R.; Su, Bing; Davis, Roger J.; Kelliher, Michelle A.  
 CS Department of Cancer Biology and Interdisciplinary Graduate Program, University of Massachusetts Medical School, Worcester, MA, USA  
 SO Molecular and Cellular Biology (2003), 23(22), 8377-8385  
 CODEN: MCEBD4; ISSN: 0270-7306  
 PB American Society for Microbiology  
 DT Journal  
 LA English  
 AB The cytokine tumor necrosis factor alpha (TNF- $\alpha$ ) stimulates the NF- $\kappa$ B, SAPK/JNK, and p38 mitogen-activated protein (MAP) kinase pathways by recruiting RIP1 and TRAF2 proteins to the tumor necrosis factor receptor 1 (TNFR1). Genetic studies have revealed that RIP1 links the TNFR1 to the I $\kappa$ B kinase (IKK) complex, whereas TRAF2 couples the TNFR1 to the SAPK/JNK cascade. In transfection studies, RIP1 and TRAF2 stimulate p38 MAP kinase activation, and dominant-neg. forms of RIP1 and TRAF2 inhibit TNF- $\alpha$ -induced p38 MAP kinase activation. We found TNF- $\alpha$ -induced p38 MAP kinase activation and interleukin-6 (IL-6) production impaired in *ripl*<sup>-/-</sup> murine embryonic fibroblasts (MEF) but unaffected in *traf2*<sup>-/-</sup> MEF. Yet, both *ripl*<sup>-/-</sup> and *traf2*<sup>-/-</sup> MEF exhibit a normal p38 MAP kinase response to inducers of osmotic shock or IL-1 $\alpha$ . Thus, RIP1 is a specific mediator of the p38 MAP kinase response to TNF- $\alpha$ . These studies suggest that TNF- $\alpha$ -induced activation of p38 MAP kinase and SAPK/JNK pathways bifurcate at the level of RIP1 and TRAF2. Moreover, endogenous RIP1 assoc. with the MAP kinase kinase kinase (MAP3K) MEKK3 in TNF- $\alpha$ -treated cells, and decreased TNF- $\alpha$ -induced p38 MAP kinase activation is observed in *Mekk3*<sup>-/-</sup> cells. These studies suggest a mechanism whereby RIP1 may mediate the p38 MAP kinase response to TNF- $\alpha$ , by recruiting the MAP3K MEKK3.

RE.CNT 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN  
 AN 2002:537202 CAPLUS  
 DN 137:260598  
 TI The death domain of NF- $\kappa$ B1 p105 is essential for signal-induced p105 proteolysis  
 AU Beinke, Soren; Belich, Monica P.; Ley, Steven C.  
 CS Division of Immune Cell Biology, National Institute for Medical Research, London, NW7 1AA, UK  
 SO Journal of Biological Chemistry (2002), 277(27), 24162-24168  
 CODEN: JBCHA3; ISSN: 0021-9258  
 PB American Society for Biochemistry and Molecular Biology  
 DT Journal  
 LA English  
 AB Stimulation of cells with tumor necrosis factor  $\alpha$  (TNF $\alpha$ ) triggers NF- $\kappa$ B1 p105 proteolysis, releasing associated Rel subunits to translocate into the nucleus and modulate target gene expression. Phosphorylation of serine 927 within the p105 PEST region by the I $\kappa$ B kinase (IKK) complex is required to promote p105 proteolysis in response to TNF $\alpha$  stimulation. In this study, the role of the p105 death domain (DD) in signal-induced p105 proteolysis is investigated. Endogenous p105 is shown to interact with the IKK complex in HeLa cells, and transient transfection expts. in 293 cells indicate that each of the catalytic components of the IKK complex, IKK1 and IKK2, can bind to p105. Interaction of p105 with both IKK1 and IKK2 is substantially reduced by deletion of the p105 DD or introduction of a specific point mutation (L841A) into the p105 DD homologous to the *lpr* mutation in Fas. Phosphorylation of immunopptd. p105 on serine 927 by purified recombinant IKK1 or IKK2 protein in vitro is dramatically reduced in both DD mutants relative to wild type. Furthermore, both of the DD mutations significantly impair the ability of low concns. of IKK2 to induce p105 serine 927 phosphorylation and proteolysis in transiently transfected 3T3

cells. However, high levels of transiently expressed IKK2 bypass the requirement for the p105 DD to induce p105 serine 927 phosphorylation. Finally, p105 serine 927 phosphorylation by the endogenous IKK complex after TNF $\alpha$  stimulation and subsequent p105 proteolysis is blocked in both p105 DD mutants when stably expressed in HeLa cells. Thus, the p105 DD acts as a docking site for IKK, increasing its local concentration in the vicinity of the p105 PEST region and facilitating efficient serine 927 phosphorylation.

RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 2001:386764 CAPLUS  
DN 135:240479  
TI Death domain signaling and its role in the central nervous system  
AU Bruce-Keller, Annadora J.  
CS Dep. Anatomy and Neurobiol., Univ. Kentucky, Lexington, KY, 40536-0298, USA  
SO Advances in Cell Aging and Gerontology (2001), 5(Programmed Cell Death, Volume I), 39-65  
CODEN: ACAGF5  
PB Elsevier Science B.V.  
DT Journal; General Review  
LA English  
AB A review with many refs. summarizes the different death receptors and the intracellular pathways they activate to induce cell life or death. The cloning and characterization of death receptors and their myriad of control mechanisms, and their roles in human physiol. and pathophysiol. are described. Topics discussed include death domain signaling components; initiation and execution of the death signal; death receptors in the central nervous system; alternative death receptor signaling; potent neuroprotective properties of tumor necrosis factor; apoptosis inhibitors; and NF- $\kappa$ B activation.

RE.CNT 148 THERE ARE 148 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 2000:790340 CAPLUS  
DN 133:355211  
TI Death domain-containing receptor 5 and compns. for treatment of immunity-related diseases, viral diseases, and cancer  
IN Ni, Jian; Gentz, Reiner L.; Yu, Guo-liang; Rosen, Craig A.  
PA Human Genome Sciences, Inc., USA  
SO PCT Int. Appl., 266 pp.  
CODEN: PIXXD2

DT Patent  
LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000066156	A1	20001109	WO 2000-US12041	20000504
	W:				
	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW:				
	GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	EP 1196191	A1	20020417	EP 2000-930329	20000504
	R:				
	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	JP 2002543151	T2	20021217	JP 2000-615040	20000504

	US 2002072091	A1	20020613	US 2001-874138	20010606
PRAI	US 1999-132498P	P	19990504		
	US 1999-133238P	P	19990507		
	US 1999-148939P	P	19990813		
	US 1997-40846P	P	19970317		
	US 1997-54021P	P	19970729		
	US 1998-42583	A1	19980317		
	US 2000-565009	A1	20000504		
	WO 2000-US12041	W	20000504		

AB The present invention relates to novel Death Domain Containing Receptor-5 (DR5) proteins which are members of the tumor necrosis factor (TNF) receptor family, and have now been shown to bind TRAIL. In particular, isolated nucleic acid mols. are provided encoding the human DR5 proteins. DR5 polypeptides are also provided as are vectors, host cells and recombinant methods for producing the same. The invention further relates to screening methods for identifying agonists and antagonists of DR5 activity, e.g., for treating graft-vs.-host disease, viral infection, cancer, and immune diseases.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN

AN 2000:617304 CAPLUS

DN 134:177079

TI The death domain of tumor necrosis factor receptor 1 is necessary but not sufficient for Golgi retention of the receptor and mediates receptor desensitization

AU Gaeta, Mary Lou; Johnson, David R.; Kluger, Martin S.; Pober, Jordan S.  
CS Department of Pediatrics, Yale University School of Medicine, New Haven, CT, 06510, USA

SO Laboratory Investigation (2000), 80(8), 1185-1194  
CODEN: LAINAW; ISSN: 0023-6837

PB Lippincott Williams & Wilkins

DT Journal

LA English

AB TNF signals are mediated through two different receptors, TNFR1 and TNFR2. In endothelial cells, TNFR1 is predominantly localized in the Golgi apparatus and TNFR2 on the plasma membrane. To investigate structural features responsible for the disparate localization, endothelial cells were transfected with epitope-tagged or green fluorescent protein-fused wild type and mutant receptor mols. Wild type receptors recapitulated the distribution of endogenous receptors. Deletions of the entire TNFR1 intracellular domain or of the C-terminal death domain (TNFR1-DD) allowed expression of the receptor on the plasma membrane. However, addition of the death domain to the C-terminus of TNFR2 (TNFR2+DD) did not lead to Golgi-retention of this chimeric receptor. Overexpressed TNFR1, TNFR2, and TNFR2+DD increased basal expression of a cotransfected NF-kB-dependent promoter-reporter gene. Overexpressed TNFR1-DD did not activate NF-kB but acted as a ligand-specific dominant neg. inhibitor of TNF actions. Unexpectedly, TNF responses were also inhibited by overexpressed TNFR1 and TNFR2+DD, but not TNFR2. We conclude that the death domain of TNFR1 is required for retention of TNFR1 in the Golgi apparatus but is not sufficient to direct Golgi retention of a TNFR2+DD chimera, and that overexpressed receptors that contain the death domain (TNFR1 and TNFR2+DD) spontaneously activate NF-kB while inhibiting TNF responses.

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1998:215327 CAPLUS

DN 129:3726

TI The death domain kinase RIP mediates the TNF-induced NF-kB signal

AU Kelliher, Michelle A.; Grimm, Stefan; Ishida, Yasumasa; Kuo, Frank; Stanger, Ben Z.; Leder, Philip

CS Harvard Med. Sch., Howard Hughes Med. Inst., Boston, MA, 02115, USA  
SO Immunity (1998), 8(3), 297-303  
CODEN: IUNIEH; ISSN: 1074-7613  
PB Cell Press  
DT Journal  
LA English  
AB The death domain serine/threonine kinase RIP interacts with the death receptors Fas and tumor necrosis receptor 1 (TNFR1). In vitro, RIP stimulates apoptosis, SAPK/JNK, and NF- $\kappa$ B activation. To define the physiol. role(s) that RIP plays in regulating apoptosis in vivo, the authors introduced a rip null mutation in mice through homologous recombination. RIP-deficient mice appear normal at birth but fail to thrive, displaying extensive apoptosis in both the lymphoid and adipose tissue and dying at 1-3 days of age. In contrast to a normal thymic anti-Fas response, rip-/- cells are highly sensitive to TNF $\alpha$ -induced cell death. Sensitivity to TNF $\alpha$ -mediated cell death in rip-/- cells is accompanied by a failure to activate the transcription factor NF- $\kappa$ B.  
RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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